



NOTES

FAT SOLUBLE VITAMINS DEFICIENCY

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Insufficient plasma concentrations of fat soluble vitamins required for normal metabolic processes

RISK FACTORS

- Reduced intake, impaired absorption, increased elimination

SIGNS & SYMPTOMS

- See individual disorders

DIAGNOSIS

- See individual disorders

TREATMENT

OTHER INTERVENTIONS

- Increased dietary intake; supplementation

VITAMIN D DEFICIENCY

osms.it/vit-d-deficiency

PATHOLOGY & CAUSES

- Body's metabolic needs not met as there is insufficient 25-hydroxyvitamin D
- Liver, kidneys hydroxylate physiologically inert vitamin D
 - Precursor molecule (7-dehydrocholesterol) exposed to ultraviolet light/dietary vitamin D → D3/D2 released into blood → first hydroxylation in liver → second hydroxylation in kidneys → metabolically active calcitriol (1,25-dihydroxyvitamin D)
- **Insufficient vitamin D** → ↓ intestinal absorption of calcium → ↓ serum calcium → ↑ serum PTH → ↑ **intestinal calcium absorption** + ↑ osteoclastic activity,

calcium resorption from bones + ↑ renal conservation of calcium → normalization of serum calcium

CAUSES

- **Insufficient** dietary, supplementary intake
- Increased need, but insufficient intake
 - Pregnancy, lactation
- Obesity, vitamin D sequestration in adipose tissue
- Impaired absorption
 - Small bowel disease; bariatric surgery; gastrectomy; pathology of hepatobiliary tree, pancreas; abetalipoproteinemia
- **Decreased synthesis in skin**
 - Insufficient sun exposure; dark skin

VISIT...

LANZAROTE
Caliente.COM

(large amounts of melanin in epidermal layer); excessive sunscreen

- Impaired liver, kidney hydroxylation
 - Cirrhosis, renal failure
- Altered vitamin D metabolism from drugs
 - Phenobarbital, phenytoin, rifampin, isoniazid, carbamazepine, ketoconazole

Genetic mutations

- **1-alpha-hydroxylase deficiency:** mutations in CYP27B1; previously called vitamin D-dependent rickets type 1A
- **25-hydroxylase deficiency:** mutations in CYP2R1; previously called vitamin D-dependent rickets type 1B
- **Hereditary resistance to vitamin D:** mutations in VDR (vitamin D receptor gene); previously called vitamin D-dependent rickets type 2

RISK FACTORS

- Risk increases with age
- Gastrointestinal tract, liver, kidney conditions
- Medications that interfere with vitamin D metabolism
- Decreased ultraviolet light exposure
 - Cold climate/high latitudes, institutionalization/incarceration

Perinatal factors (↓ neonatal vitamin D stores)

- Exclusively breastfed infants
 - Maternal vitamin D status dictates amount vitamin D in milk
- Premature birth
 - Low stores of vitamin D
- Low maternal vitamin D during gestation

COMPLICATIONS

- Related to accompanying hypocalcemia
 - Osteoporosis
 - Increased risk of fractures
 - Osteomalacia
 - Rickets (children)
 - Secondary hyperparathyroidism

SIGNS & SYMPTOMS

SIGNS & SYMPTOMS

- Mild deficiency may be asymptomatic
- Decreased bone density, osteoporosis
- Fractures
- Dental enamel hypoplasia
- Severe deficiency
 - Hypocalcemia-related osteomalacia symptoms
 - Bone tenderness/pain
 - Muscle weakness, cramping, numbness/tingling, positive Trousseau sign, Chvostek's sign
 - Bone malformations: difficulty ambulating, waddling gait

DIAGNOSIS

DIAGNOSTIC IMAGING

X-ray

- High-stress areas
 - Fractures; vertebral compression fractures
- Radiolucent bands (indicate pseudofractures)
- Codfish vertebrae
 - Biconcave vertebral discs
- Children
 - Epiphyseal plate widening; osteopenic/malformed long bone shafts, pathological fractures

Dual-energy X-ray absorptiometry (DEXA)

- Decreased bone density

LAB RESULTS

- Decreased serum 25-hydroxyvitamin D (calcidiol) level
- Elevated alkaline phosphatase (bone turnover marker)
- Decreased serum calcium
- Increased parathyroid hormone (PTH)

TREATMENT

OTHER INTERVENTIONS

- Vitamin D3 supplementation
 - Dietary: fish, egg yolk, fortified foods
- Supplementation
- Ultraviolet light/natural sunlight exposure
- Increase calcium intake

VITAMIN K DEFICIENCY

osms.it/vit-k-deficiency

PATHOLOGY & CAUSES

- Body's metabolic needs not met; insufficient vitamin K
 - Coagulation function
 - Bone biology
 - Vascular biology
- Dietary vitamin K1 (phylloquinone) → bile salts make fat soluble vitamin soluble → incorporated into gastrointestinal tract's micelles → absorbed by small intestine → integrated into chylomicrons → transported to portal circulation → liver uses to synthesize coagulation factors, other essential proteins
- Diseases of small intestines, liver, gallbladder, pancreas

RISK FACTORS

Infants

- No vitamin K1 prophylaxis at birth
- Immature liver uses vitamin K inefficiently
- Low vitamin K stores
- Sterile gut
- Maternal ingestion of coumarin-like anticoagulants/some anticonvulsants/antibiotics during gestation
- Antibiotic administration (destroys developing gut flora)

Adults

- Prolonged diarrhea
- Use of broad-spectrum antibiotics, low intake of vitamin K
- TPN administration without added vitamin K

COMPLICATIONS

- Most common
 - Bleeding, ranging from mild (mucocutaneous) to severe (intracranial hemorrhage most common in late onset VKDB)
- Impaired bone mineralization
- Vascular calcium deposits

TYPES

Infancy: vitamin K-deficiency bleeding (VKDB)

- Early-onset VKDB: occurs within first 24 hours of life; caused by immature liver function, low stores of vitamin K at birth, sterile gut, exclusive breastfeeding (breastmilk low in vitamin K), medications present in maternal circulation interfering with vitamin K (anticonvulsants, warfarin)
- Classic VKDB: occurs between 1–4 weeks; prevented by vitamin K1 prophylaxis at birth
- Late-onset VKDB: occurs between 3 weeks–8 months; associated with lack of vitamin K1 prophylaxis at birth, exclusive breastfeeding (breastmilk low in vitamin K)

Later childhood, adulthood

- Fat absorption and vitamin K metabolism disorders

SIGNS & SYMPTOMS

- Low bone density signs

Impaired coagulation

- *Mucocutaneous bleeding*: gingival, nasal, easy bruising
- *Gastrointestinal bleeding*: melena
- *Genitourinary bleeding*: hematuria
- *Neonatal bleeding*: umbilical stump/circumcision site
- *Intracranial hemorrhage*: vomiting, seizures

DIAGNOSIS

DIAGNOSTIC IMAGING

DEXA

- Low bone mineralization

LAB RESULTS

- Coagulation studies
- Prolonged prothrombin time (PT), partial thromboplastin time (PTT), International Normalized Ratio (INR)
- Elevated serum undercarboxylated proteins (proteins induced by vitamin K absence)

TREATMENT

OTHER INTERVENTIONS

- Administer Vitamin K
 - Subcutaneous phytonadione
- ↑ dietary vitamin K
 - Liver, green leafy vegetables (broccoli, spinach, kale)